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EXAMINER	
PETERSEN, CLARK D	
ART UNIT	PAPER NUMBER
1657	

SHORTENED STATUTORY PERIOD OF RESPONSE	NOTIFICATION DATE	DELIVERY MODE
3 MONTHS	01/08/2007	ELECTRONIC

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Notice of this Office communication was sent electronically on the above-indicated "Notification Date" and has a shortened statutory period for reply of 3 MONTHS from 01/08/2007.

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Office Action Summary	Application No.	Applicant(s)	
	10/606,162	REMACLE ET AL.	
	Examiner	Art Unit	
	Clark D. Petersen	1657	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 1 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *13 September 2006*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,4,7-9 and 11-36* is/are pending in the application.

4a) Of the above claim(s) *16-21 and 30-36* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,4,7-9,11-15 and 22-29* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

The text of those sections of Title 35 U.S. Code not included in this action can be found in a prior Office Action.

All objections and rejections not repeated in the instant Action have been withdrawn due to applicant's response to the previous action.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1 and 15 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention. Enablement is considered in view of the Wands factors (MPEP 2164.01 (A)). These include: nature of the invention, breadth of the claims, guidance of the specification, the existence of working examples, state of the art, predictability of the art and the amount of experimentation necessary. All of the Wands factors have been considered with regard to the instant claims, with the most relevant factors discussed below.

Nature of the invention: Claim 15 is drawn to a method of evaluating cell activation by evaluating the phosphorylation state of specific, named proteins from the list of Table I in the specification. Further, the claim is restricted according to the restriction requirement sent March 27, 2006 and the responsive election filed May 2, 2006, to the determination of cell activation by measuring phosphorylation of CDK8, PAK6, and MKK3.

Breadth of the claims: The claims are narrow in that applicant proposes to measure an aspect of cell activation by studying three cellular proteins in particular, namely CDK8, PAK6, and MKK3.

Guidance of the specification and existence of working examples: The specification provides guidance generally in determining the level of phosphorylation of a protein, or a cascade of proteins, in a comparison of activated versus untreated cells. Specific examples are given in the working examples of experiments performed with Akt, Erk, p38, as a few examples. However no working examples are given for determining phosphorylation status of CDK8, MKK3, or PAK6, and mention of these kinases is absent elsewhere in the specification, other than in Table 1 which provides guidance for the claims.

Predictability and state of the art: Studying phosphorylation of cellular proteins is a well-accepted method of studying aspects of cell physiology. It is reasonable to characterize the phosphorylation of members of signaling cascades along with changes in cellular dynamics. However CDK8 is unique among CDKs in that it appears that its activation is not dependent on phosphorylation as is observed with other CDK family

members. Hoeppner et al (2005) determined that CDK8 is lacking the threonine in its activation loop that is present in other CDKs, and is phosphorylated to activate such CDKs. Rather CDK8 is activated by interaction of the residue that replaces the otherwise-phosphorylated threonine with its binding partner Cyclin C (see Possible mechanisms of CDK8 Activation, pp. 839-840, for example).

Therefore, no evidence is provided in either the instant specification for determination of activation of CDK8 by phosphorylation, and there is evidence in the literature that phosphorylation of CDK8 is not an effective means of determining cellular activation state.

Amount of experimentation necessary: As discussed above, the instant specification describes generally a method of characterizing cellular physiological responses to activating stimuli by measuring phosphorylation of components of cellular signaling cascades. It provides working examples for measuring phosphorylation of members of a few pathways including Akt, ERK, and p38, among others. However the instant specification does not provide working examples of phospho-specific antibodies for the elected species CDK8 and PAK6 and it is therefore not detailed enough to allow one of ordinary skill in the art to perform a cell activation assay using phosphorylation of these signaling molecules as a readout.

In view of the lack of guidance from the specification, the absence of working examples, literature that undermines the assertions of the instant application, the skilled artisan would have required an undue amount of experimentation to make and/or use the claimed invention. Therefore claims 1 and 15, as they are drawn to the elected

species of the combination of CDK8, PAK6, and MKK3, are not considered to be enabled by the instant specification.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1, 4, 7, 8, 11, 12, and 14 are rejected under 35 U.S.C. 102(b) as being anticipated by Schooler et al (Anal Biochem, 2000). Schooler et al teach a ratiometric assay of epidermal growth factor receptor tyrosine kinase activation. They teach the measurement of EGFR activation in the MDA-MB-468 cell line (see p. 136, col. 2, for example). They teach that they treat the cells with purified epidermal growth factor, reading on 26 and 27 (see p. 136, col. 2, for example). They teach that 96 well plates, which fit the definition of plastic solid supports, are coated with anti-EGFR antibody, a capture molecule. Cell lysates - treated or untreated with EGFR – are added to the wells and incubated so the antibody captures EGFR in the lysates. The plates are washed, and phosphotyrosine antibody is added to half the wells while general EGFR antibody, which detects EGFR regardless of phosphorylation state, is added to the other half of the wells on each plate (see "Tyr(P)/EGFR ELISA", pp. 136-137, for example). By this method they can determine the ratio of phosphorylated to total EGFR in response to treatment with EGF. They teach that their assay is a rapid, sensitive, and

reliable means to assess receptor tyrosine kinase activity in all cell types (see p. 141, col. 2, for example).

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 1, 4, 7-9, 11, 12, 14, 22-27, and 29 are rejected under 35 U.S.C. 103(a) as being unpatentable over Schooler et al (Anal Biochem, 2000) in view of Paweletz et al (Oncogene, 2001).

This is a new rejection necessitated by applicants' amendment filed 13 September 2006.

The teachings of Schooler et al are discussed above and applied as before.

Additionally Schooler teaches a method of detecting phosphorylation of the EGFR in response to EGF administration, reading on a chemical treatment as recited in claim 27.

Schooler et al do not expressly teach that first and second arrays are present on different supports, and that different populations of cells are assayed and compared.

Paweletz et al teach a method of microdissecting distinct cell populations, from within and from around a tumor, from a single patient. They collect cellular lysates and immobilize them on slides made of nitrocellulose and glass. They analyze the

phosphorylation state of Akt and ERK, for example, and correlate it with tumor progression, which reads on evaluating an activated state of cells (see Results, p. 1985, Fig. 5, p. 1986, and Materials and Methods pp. 1987-1988, for example). Paweletz et al also teach that multiple slides can be employed in their method, some slides treated as negative controls while separate slides are treated as experimental slides (see p. 1982, col. 2, for example). It is known in the art that cancer derives from accumulated genetic mutations, reading on claim 24. Because cancer is a disease in which cells dedifferentiate to some degree, the teachings of Paweletz et al read on claim 25.

A person of ordinary skill in the art at the time the invention was made would have been motivated to capture phosphorylated proteins in an array by specific epitope binding capture molecules immobilized on the array, because Paweletz et al teach that observing the phosphorylation state of multiple proteins in parallel yields a signature of diseased tissue, and Schooler et al teach that one can observe proteins on a solid support in a sensitive, reliable way by capturing phosphorylated proteins on a solid support with immobilized epitope-binding capture molecules and then using phosphospecific antibodies to detect phosphorylation status, and that this method should be amenable to many different signaling molecules.

Hence, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to immobilize multiple proteins on a solid support array through the use of epitope-binding capture molecules followed by detection of total versus phosphorylated proteins.

Claims 1, 4, 7-9, 11-15, 22-27, and 29 are rejected under 35 U.S.C. 103(a) as being unpatentable over Schooler et al (Anal Biochem, 2000) in view of Paweletz et al (Oncogene, 2001) and in view of Lee et al (2001).

This is a new rejection necessitated by applicants' amendment filed 13 September 2006.

The teachings of Schooler et al are discussed above and applied as before.

The teachings of Paweletz et al are discussed above and applied as before.

Paweletz et al and Schooler et al do not expressly teach study of phosphorylation status of a transcription factor or MKK3.

Lee et al teach that it is possible to activate cells with a chemical treatment, in this case a transfection mixture comprising a transfectable plasmid for β-pix, which leads to phosphorylation of MKK3 and the transcription factor ATF-2. They demonstrate that they can determine the ratio of phosphorylated MKK3 and ATF-2 to unphosphorylated MKK3 and ATF-2 with an antibodies specific to phospho-MKK3 and phospho-ATF2, and use it measure activation of these proteins in cells activated with the β-Pix transfection mixture vs. untreated cells (see Materials and Methods, pp.25067-25068, and see Fig. 5, p. 25069, as examples).

A person of ordinary skill in the art at the time the invention was made would have been motivated to measure phosphorylation of a transcription factor in a method of measuring cell activation, because Lee et al teach that transcription factors such as ATF2 are targets of kinases such as p38 which respond to activating signals such as

growth factors (see Fig. 3, p. 25068, for example), and that MKK3 also responds to stimuli which mimic activating growth factors (see Fig. 5, p. 25069, for example).

Hence, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to study the phosphorylation status of a transcription factor, as well as MKK3, in a method of studying cell activation.

Claims 1 and 4 are rejected under 35 U.S.C. 103(a) as being unpatentable over Schooler et al in view of Paweletz et al, and further in view of Gustafson et al (US Patent # 5413939, issued 5/9/1995).

This is a new rejection necessitated by applicants' amendment filed 13 September 2006.

The teachings of Schooler et al are discussed above and applied as before.

The teachings of Paweletz et al are discussed above and applied as before.

Paweletz et al and Schooler et al do not expressly teach that metals, compact discs and electronic devices can be used in a method of testing cell activation.

Gustafson et al describe a method of binding antigens or antibodies to a compact disc, adding the complementary antigen or antibody, and then testing interferometrically the binding of antigen to antibody. Gustafson et al also teach that metals can be used for immobilizing protein with the expectation of measuring binding properties of complementary molecules (see col. 1, lines 29-36).

A person of ordinary skill in the art at the time the invention was made would have been motivated to use a metal surface, or a compact disc, and do measurements

electronically in a method of determining cell activation by measuring binding of phosphospecific antibodies to immobilized phosphoproteins, because Gustafson et al teach that one can bind proteins to a metal surface or compact disc, and then efficiently test binding of antibodies to the immobilized proteins, in the instant situation using the technique of interferometry. One would have been motivated to do so, because Gustafson et al teach that their system is characterized by high linearity, a large dynamic range, a background free output, few process steps, short incubation times, and low coefficient of variation in relation to a standard measurement (see col. 2, lines 10-29, for example). Gustafson et al also teach that immobilizing proteins on metal surfaces allows light to reflect, making possible detection of changes in wavelength of that reflected light which can be correlated with binding of an analyte, such as an antibody.

Hence, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to was made to use a metal surface, or a compact disc, and do measurements electronically in a method of determining cell activation by measuring binding of phosphospecific antibodies to immobilized phosphoproteins.

Claims 1, 4, 7-9, 11, 12, 14, 22-27, and 29 are rejected under 35 U.S.C. 103(a) as being unpatentable over Schooler et al in view of Paweletz et al and further in view of Chang et al (USPGPub US2002/0192654 A1, filed 6/15/2001).

This is a new rejection necessitated by applicants' amendment filed 13 September 2006.

The teachings of Schooler et al are discussed above and applied as before.

The teachings of Paweletz et al are discussed above and applied as before.

Paweletz et al and Schooler et al do not expressly teach the use of silicon as a solid support for phosphoproteins in a method of determining cell activation.

Chang et al teach that it is well known in the art to use silicon as a bio-chip substrate for DNA or protein immobilization (see Page 1, paragraph 0002, for example). They teach that the concept of the biochip was developed in the late twentieth century, and that the biochip is broadly defined as a product for immobilizing DNA, protein, or cell structures on a silicon for biochemical analysis.

A person of ordinary skill in the art at the time the invention was made would have been motivated to use a silicon support in a method of measuring phosphoproteins for the purpose of determining cell activation, because determining cell activation depends on immobilizing phosphoproteins on a solid support, and Chang et al teach that silicon is useful as a support for immobilized proteins because it is a material associated with high reliability, and rapid and accurate analysis (see para [0002], for example).

Hence, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to immobilize capture molecules on silicon supports in a method of studying cell activation.

Claims 22 and 28 are rejected under 35 U.S.C. 103(a) as being unpatentable over Schooler et al in view of Paweletz et al and in view of Matsui et al (EMBO J, 1996).

This is a new rejection necessitated by applicants' amendment filed 13 September 2006.

The teachings of Schooler et al are discussed above and are applied as before.

The teachings of Paweletz et al were discussed above and are applied as before.

Paweletz et al and Schooler et al do not expressly teach the treatment of cells after lysis with a compound, and testing the response of kinase activity within the lysate to a test compound.

Matsui et al teach a method of lysing cells and concentrating a particular kinase activity from the cell lysate. They then provide various cellular proteins as substrates for the kinase activity. They demonstrate that adding a test compound to the kinase changes its activity toward cellular protein substrates; in this case, addition of GTPγS changes the phosphorylation activity of the kinase complex towards its substrates S6 protein, Protein Kinase C, and Myelin Basic Protein (MBP) (see Materials and Methods, p2214-2215, and Fig. 4, p. 2210, as examples).

A person of ordinary skill in the art at the time the invention was made would have been motivated to use the method of Matsui et al of isolating lysate-kinase activity first, and then applying a test compound, in the method of testing phosphorylation equilibrium by binding phosphoproteins to a solid support, because Matsui et al demonstrate that it is possible to measure a change in phosphorylation status of a substrate by stimulating a kinase activity after cells have already been lysed, and Matsui et al demonstrate that one can concentrate kinase activity and thus have a stronger readout.

Hence, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to lyse cells before treating them with a compound in a method of studying cell activation.

Response to Arguments

Applicants' arguments filed 13 September have been fully considered but are not deemed persuasive.

Applicants traverse the rejection of claims 1 and 15 under the first paragraph of 35 USC 112 as failing to comply with the enablement requirement.

Applicants' arguments have been fully considered but they are deemed unpersuasive.

Applicants argue that CDK8 is the largest CDK known so far. It harbors an extended C-terminal tail which contains serine and threonine residues; furthermore, it shares some similarities with CDK9, which exhibits autophosphorylation in situations of activation.

The argument that studying CDK8 phosphorylation by comparison with CDK9 enables the instant application is not deemed persuasive. Regarding the comparison of CDK8 to CDK9, Garber et al (Mol Cell Biol, Sept. 2000) acknowledge they have not fully elucidated the exact extent and location of phosphorylation necessary in the C-terminal tail to activate CDK9 in a physiologically important manner (see p. 6963, bottom of column 1, for example). They teach that "this peptide contains several possible Ser and Thr phosphorylation sites....the radioactive sequencing experiment did not exclude

possible phsophorylation at the other residues within this peptide, and at least two additional peptides were labeled less extensively; we have not identified these minor sites of CDK9 autophosphorylation". To generate a phosphospecific antibody, it is well known to those of ordinary skill in the art that at the minimum specific peptides containing specific, synthetically phosphate-labeled residues must be injected in a host animal to generate an antibody response. Applicants have not provided this minimal information. Furthermore, it is well known in the art that generating antibodies is not an exacting process; some peptides do not generate a sufficient response in host animals to allow purification of a useful antibody.

Furthermore, it is evident from the articles provided by applicant that the functions of CDK8 and CDK9 are not exactly the same. An included document (Gold et al, Nucleic Acids Res, 1998) does not show which residues in CDK8 are potentially phosphorylated in the events of cell activation. Furthermore, Gold et al imply it is not clear how CDK8 phosphorylation, if any exists, defines cell activation. For example, they teach:

> Two CDKs have now been identified, CDK8 and CDK9, that appear to be able to function after initiation to activate processivity of the RNAPII complex. The existence of these two and perhaps additional kinases therefore allows a layer of regulation to control productive transcription of mRNA genes. CDK8 is a component of the RNAPII holoenzyme and, therefore, CDK9 may need to be specifically recruited to the holoenzyme to activate elongation. A major challenge for future understanding of control of transcriptional elongation will be to identify and elucidate mechanisms whereby CDK8 function is regulated in the holoenzyme and how CDK9 function is recruited to the holoenzyme."

Therefore, the included literature states that it is not known what mechanisms regulate CDK8 function. For all the reasons stated above, it is incumbent upon the applicants to include a working example describing how CDK8 phosphorylation is measured, how the reagents for detecting this phosphorylation are obtained, and how phosphorylation of CDK8 reads upon an activation state. For these reasons, the rejection under 35 USC 112 first paragraph, for reasons of lack of enablement, is maintained.

Applicants also argue that PAK6 is regulated by phosphorylation and its phosphorylation state may well inform about the activation status of target cells. However the reference provided by applicant provides that the functions of PAK6, as well as regulation of its functions, are not elucidated. For example, Jaffer et al (2002) state that "differences imply that Group I and II Paks may be regulated differently and may have different downstream effectors" (see p. 714, col. 2). They also teach that "observations indicate that we need to identify physiologically relevant substrates of the Group II Paks before we can draw firm conclusions about their activity" (see p. 715, bottom of col. 1). In the context of this passage, Jaffer et al teach that the relevance of phosphorylation events, where and whether they occur, as well as how they can be related to overall cell activation, are not known. For these reasons, the rejection under 35 USC 112 first paragraph, for reasons of lack of enablement, is maintained.

Applicants traverse the rejection of claims 1, 2, 11, 12, 14, and 22-25 under 35 USC 102(b) as being anticipated by Paweletz et al (2001). Applicants argue that Paweletz et al do not teach contacting a biological sample with two arrays, wherein a first array is used to immobilize only phosphorylated cellular proteins, while the second array is used to immobilize the proteins regardless of their phosphorylation state, and then quantifying the level of phosphorylation of said immobilized proteins by measuring the signal ratio between the phosphorylated versus the total proteins present in the sample. Furthermore, it is argued, Paweletz does not teach that the target molecules are bound to the capture probes via sites or epitopes not bearing a phosphate moiety, and therefore do not teach all the limitations of the currently amended claim 1, and do not anticipate currently amended claims 1, 2, 11, 12, 14, and 22-25.

In light of applicants' amendment to claim 1, the rejection of claims 1, 4, 11, 12, 14, and 22-25 as being anticipated by Paweletz et al (2001) is withdrawn. However, note the claims 1, 4, 7-9, 11, 12, 14, 22-27, and 29 have been rejected under the new rejection under 103(a) discussed above.

Applicants traverse the rejection of claims 1-9, 11, 12, 14, 15, 22-27, and 29 under 35 USC 103(a) as being unpatentable over Paweletz et al (2001) in view of Huang (2001).

In light of applicants' amendment to claim 1, the rejection of claims 1-9, 11, 12, 14, 15, 22-27, and 29 as being unpatentable over Paweletz et al (2001) in view of Huang (2001) is withdrawn.

Applicants traverse the rejection of claims 1-4 under 35 USC 103(a) as being unpatentable over Paweletz et al (2001) in view of Huang (2001) and further in view of Gustafson et al (US 5,413,939) or Chang et al (US2002/0192654A1).

In light of applicants' amendment to claim 1, the rejection of claims 1-4 as being unpatentable over Paweletz et al (2001) in view of Huang (2001) and further in view of Gustafson et al (US 5,413,939) or Chang et al (US2002/0192654A1) is withdrawn.

Applicants traverse the rejection of claims 22 and 28 under 35 USC 103(a) as being unpatentable over Paweletz et al (2001) in view of Matsui et al (1996).

In light of applicants' amendment to claim 1, the rejection of claims 22 and 28 as being unpatentable over Paweletz et al (2001) in view Matsui et al (1996) is withdrawn.

Conclusion

No claims are allowed.

Applicants' necessitated the new grouns of rejection presented in this office action. Accordingly, **THIS ACTION IS MADE FINAL.** Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the

shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Clark D. Petersen whose telephone number is (571)272-5358. The examiner can normally be reached on M-F 8:30-5:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jon Weber can be reached on (571)272-0925. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

CDP
12/14/2006

JON WEBER
SUPERVISORY PATENT EXAMINER